to the DNA and activates certain genes (Behrens, J. et al., Nature 382, 638 – 642, 1996).

This signal path also plays an important role in the formation of tumors. In epithelial cells of the colon, the cytoplasmic pool of ß-catenine is strictly regulated by the tumor suppressor gene product APC (Adenomatosis Polyposis Coli). Mutations of APC, such as those occurring in 80% of all colon cancers, lead to shortened forms of the APC protein, which are no longer able to destabilize ß-catenine. As a result, permanent complexes of ß-catenine with the HMG transcription factor TCF-4, which are asserted to be responsible for the transformation of the cells, are found in these tumors. This theory is supported by the recent finding that, in tumors in which APC is not changed, mutations of ß-catenine occur. These also lead to cytoplasmic stabilization of ß-catenine and to an association with LEF-1/TCF factors (Morin, P.J. et al., Science 275, 1787 – 1790).

Brief description of the drawing

The invention is disclosed below with reference being had to the drawing, wherein

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Fig. 1 is the amino acid sequence 1 to 840 of ~~conductine~~ Conductin (SEQ ID NO:1);

Fig. 2 is the nucleotide sequence of ~~conductine~~ Conductin (SEQ ID NO:6) (SEQ ID NO:1);

Fig. 3 is the gene comparison sequence and the nucleotide sequence (nucleotide # 215 to 2737 of SEQ ID NO:6); and

Fig. 4 is a showing of of interaction stuidies in the 2-hybrid system.

Description of the invention

It is an object of the present invention to find a new way to prevent the formation of tumors. It is based on the objective of finding a method for controlling the regulation of ß-catenine in cells of the body.